EXHIBIT 5

EXPERT REPORT BY DR. ALAN C. WHITEHOUSE

A. Qualifications.

1. I am Dr. Alan C. Whitehouse. My address is 28810 North Milan Road, Chattaroy, WA 99003.

2. I am licensed in Washington and Montana. I am Board Certified in pulmonology and internal medicine. I currently practice chest medicine at the Center for Asbestos Related Disease (CARD) in Libby, Montana, where we have over 1,800 active cases of asbestos disease from exposure to Libby asbestos. I have practiced pulmonary medicine in Spokane, Washington, from 1969 - 2004, and in Libby, Montana, from 2004 to date.

3. My curriculum vitae is attached as Exhibit 1.

4. In addition, I have been an invited speaker on the subject of Libby asbestos disease at various locations across the country. See Exh. 1, Invited Presentations on Asbestos Disease.

5. Since 1980 I have evaluated or treated over 700 patients for asbestos disease from Libby asbestos. I have consulted with CARD physicians on many others. Since 1980 I have also evaluated or treated over 500 patients with asbestos disease from predominately chrysotile exposures. I am in a position to compare asbestos disease from Libby asbestos to asbestos disease from chrysotile asbestos. Chrysotile asbestos

is the usual form of asbestos used in building materials in the United States, accounting for about 95% of the total asbestos used in the United States. Fraser and Pare text, p.2420.

6. I have treated the entire range of pulmonary diseases. In my practice in Spokane in the years 1994-2004, the majority of my time, probably about 90%, was related to general chest diseases, including asthma, emphysema, lung cancer and the care of hospitalized patients. About 5-10% of my time was spent on asbestos related issues and other pneumoconioses. Probably about 10% of my time was related to industrial disease. Currently I spend a small amount of time on legal matters, but for the most part, my time is devoted to patient care and research.

7. In 30 years of practice I have probably testified at trial 10-15 times, about half for the plaintiff and half for the defendant. I testified in three asbestos trials relating to exposure from the W.R. Grace mine and mill near Libby, and one trial on the same subject in Missoula, Montana. These trials related to asbestos disease from Libby asbestos. In addition, my deposition has been taken on the subject of asbestos disease probably 25-30 times. I have testified in five Libby asbestos cases before the Montana Workers' Compensation Court.

8. I have published a paper on asbestos disease in Libby, titled

"Asbestos-Related Pleural Disease Due to Tremolite Associated with Progressive Loss of Lung Function: Serial Observations in 123 Miners, Family Members, and Residents of Libby, Montana," Am J Ind Med 46:219-225 (2004). A copy of the paper is attached as Exhibit 2. 123 patients were followed for an average of 35 months. Lung function was measured in terms of total lung capacity, forced vital capacity and diffusion capacity. The range of loss was 2-3% per year for each of these lung functions.

9. I have published a paper titled Whitehouse et al (2008), "Environmental Exposure to Libby Asbestos and Mesotheliomas." Eleven cases are discussed. A copy of the paper is attached as Exhibit 3.

10. Over the last three decades I have practiced occupational medicine. I have performed studies for companies, done screenings for companies and done disability exams for companies. In the 1980s I was involved in multiple asbestos disease screening programs for companies. I have also done independent medical examinations for the State of Washington Department of Labor and Industry for decades.

B. The mechanism for asbestos disease.

11. Asbestos is a mineral fiber. There are two kinds, serpentine (chrysotile) and asbestiform amphiboles. Chrysotile asbestos is the kind most often used commercially in building products. Chrysotile asbestos is

more curly, or more club-like, whereas amphibole asbestos is like tiny needles or spears. Libby asbestos is an amphibole. It has generally been referred to as tremolite. Using state of the art methodology, Meeker (2003), p.1959, has determined that the Libby asbestos is approximately 84% winchite, 11% richterite and 6% tremolite. Meeker, Figure 6 shows the incidence and close chemical form relation as between winchite, tremolite and richterite. Meeker, p.1967, suggests that:

> The Vermiculite Mountain amphibole asbestos could, for the purposes of regulation only, be considered equivalent to tremolite or soda-tremolite asbestos in accordance with current and past industrial terminology for the Vermiculite Mountain amphiboles.

12. In relative terms of their length to width (aspect ratio), Libby asbestos fibers are long and sharp, like needles. The fibers breathed in are microscopic, as are the alveoli (tiny air sacs) in the lungs. When breathed in, the fibers lodge in the structure around the alveoli, and are too small to be expelled. Asbestos fibers irritate and inflame the lung tissue structure around the air sacs (the interstitia). Scarring in the interstitia is interstitial disease. When the interstitia are significantly scarred, they can no long expand or contract fully, and breathing is restricted.

13. The amphibole fibers also migrate to the outside portion of the

lung, where they scar and inflame the pleura (the lung lining) and cause pleural disease. See the Frazer and Pare text, p.2809. Pleural disease seems particularly pronounced with Libby asbestos fibers.

14. The normal pleura is actually thinner than a blown up balloon. It is a very thin membrane, and it can expand like a balloon. Asbestos fiber scarring causes the pleura to look much like the orange portion of an orange rind, and can be just as thick. When surgeons peel it off the pleura, they call it a rind. When the lung lining becomes as thick as an orange rind, it can no longer expand freely and breathing is restricted. Asbestos disease is generally a restrictive lung disease.

C. Diagnosis of Asbestos-Related Disease.

15. For the diagnosis of asbestos related disease, we use the criteria of the American Thoracic Society (2004) Official Statement, "Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos," Am J Respir Crit Care Med, 170:691-715. Asbestos interstitial disease is due to scarring in the lung structure around the alveoli (air sacs) from the poking and inflammation from asbestos fibers. The asbestos pleural disease seen in Libby is due to the scarring and inflamation in the pleura (the lung lining) from asbestos fibers. The CARD Clinic generally uses "ARD" (asbestos related disease) to refer to nonmalignant asbestos disease. Sometimes the

term "asbestosis" is used as an umbrella term, covering asbestos interstitial disease and asbestos pleural disease, since they are essentially the same disease process. The Rosenstock text, p.374, states: "Some investigators have used the term asbestosis to encompass nonmalignant asbestos-related pleural abnormalities."

16. ATS Official Statement (2004) states the diagnostic criteria as follows:

> Evidence of structural pathology consistent with asbestos-related disease as documented by imaging or histology.
>
> Evidence of causation by asbestos as documented by the occupational and environmental history, markers of exposure (usually pleural plaques), recovery of asbestos bodies, or other means.
>
> Exclusion of alternative plausible causes for the findings.

The ATS states that "the occupational history should emphasize occupational and environmental opportunities for exposure that occurred about 15 years or more before presentation."

17. I have taken hundreds of work histories relating to asbestos exposure at the W.R. Grace mine and mill near Libby, Montana, and am familiar with conditions in the various jobs there. I have also taken hundreds of histories of exposure from family members of workers and Libby community members. Pathways for asbestos disease from Libby asbestos

exposure are discussed at Peipins et al (2003). I have also evaluated hundreds of patients concerned about asbestos disease from Libby exposures, and have concluded that they do not have asbestos disease. For scores of these same patients, I have diagnosed lung disease other than asbestos disease.

18. Asbestos disease causes a restrictive defect. The amount of air breathed in is restricted. The physical examination includes determinations of chest restriction, the presence of rales (the crackling sound of scarred air sacs reopening), and an evaluation of shortness of breath. While chest x-rays occasionally show abnormalities not seen on CT scan, chest x-rays often miss parenchymal abnormalities of asbestosis seen on CT scan. See the Fraser and Pare text, p.2431. Chest x-rays miss an even higher percentage of pleural abnormalities, as compared to CT scans. *Id.,* pp. 2431 and 2440. ATS Official Statement (2004), p.696, states: "Only 50 to 80% of cases of documented pleural thickening demonstrated by autopsy, conventional CT or high resoltuion CT (HRCT) are detected by chest radiograph (42, 43)." Frequently we see subpleural interstitial fibrosis on CT scans which is often not seen on chest x-ray, and which may play a significant role in the severity of the disease process. See the Schwarz and King text, p.422; ATS (2004) Official Statement, p.702.

19. At our clinic, lung function tests are performed in accordance with ATS criteria. We generally use Knudson norms for vital capacity (spirometry), Intermountain Thoracic Society for lung volumes, and Miller for diffusion capacity. Crapo norms are used as required for AMA Guides disability determinations. Abnormal values are under 80% of predicted or over 120% of predicted.

20. Pulmonary function tests are the measure of severity of asbestos disease. The Rosenstock text, p.370, states: "Pulmonary function tests are the most important tool for the functional assessment of nonmalignant asbestos-related effects." Of all lung function tests, the three most important in asbestos disease are forced vital capacity (FVC), total lung capacity (TLC) and diffusion capacity (DLCO). The Fishman text, p.950, states "[t]he characteristic pulmonary function changes of asbestosis are a restrictive impairment with a reduction in lung volumes (especially FVC and total lung capacity) decreased diffusion capacity, and arterial hypoxemia." ATS (2004) Official Statement, adopts the above quote at p.697. The AMA Guides to the Evaluation of Permanent Impairment (5th Ed), p.107, "considers only pulmonary function measurements for an impairment rating."

21. There are three components to pulmonary function tests. First is spirometry, which measures the amount of volume of the lung and the

rapidity of inhalation, which gives an index of air flow and lung volumes. We usually do this before and after brochodilator.

Second, we do lung volumes in what is called a body box, or plethysmograph, where we measure very small changes in air flow, pressure and volume, with a shutter and a closed system. Using Boyle's law, one can calculate the volume of the lung.

Third, we measure diffusion capacity, by having the patient breathe a small percentage of carbon monoxide, using very tiny tracer amounts of methane, which is not absorbed, and we measure what comes out of the lungs. We measure the methane, measure the carbon monoxide, and the differential uptake gives us the carbon monoxide diffusion capacity. Diffusion capacity is the efficiency of the lungs in transferring oxygen into the blood stream. In restrictive lung disease there is interference with the air/blood interface due to the increased scarring that forms a barrier between the blood vessels and the alveoli (air sacs).

22. When asbestos disease due to Libby asbestos exposure is first diagnosable, there usually are no symptoms, only positive findings on chest x-ray or CT. The disease may take decades to progress to a point of severity. Severe disease may include shortness of breath, chest pain, rales, clubbing of the fingernails, hypoxia, cor pulmonale, pleural effusions, and

oxygen dependency. See ATS (2004) Official Statement. At end stage the patient is bedridden, oxygen dependent, and generally the hypoxia will lead to organ malfunction and death.

D. Pleural Disease Generally.

23. "Pleural plaques" are a lesion of the parietal pleura, typically presenting circumscribed borders and a "raised straight surface with clear cut edges when seen face on." ATS (2004) Official Statement, p.704.

24. The ATS (2004) Official Statement, p. 705, literature review collects many studies showing that pleural plaques are associated with decrements in lung function:

> Studies of large cohorts have shown a significant reduction in lung function attributable to the plaques, averaging about 5% of FVC (forced vital capacity), even when interstitial fibrosis (asbestosis) is absent radiographically (74, 76, 107). The presence of circumscribed plaques can be associated with restrictive impairment and diminished diffusing capacity on pulmonary function testing, even in the absence of radiographic evidence of interstitial fibrosis (108, 109).

Textbook authors concur with the ATS (2004) Official Statement. *Fishman's Pulmonary Diseases and Disorders* (4th Ed 2008), p.945, states: "pleural disease has been recognized as the cause of reduced pulmonary function since the 1970s." The Fishman text, p.945, notes studies showing pleural plaques associated with decrements in FVC and FEV_1. *Fraser and*

Pare's Diagnosis of Diseases of the Chest (4th Ed. 1999), p.2446, states: "it has become clear that asbestos related pleural disease can also affect lung function adversely (718, 721, 737, 738). Both pleural plaques and diffuse pleural thickening cause decreases in vital capacity, although the effects of diffuse thickening are more marked." Rosenstock et al, *Textbook of Clinical, Occupational and Environmental Medicine* (2nd Ed. 2005), p.372, states: "studies of asbestos exposed populations have variably observed small reductions in ventilation capacity associated with plaques."

25. Importantly, the ATS (2004) Official Statement, p.705, notes that in patients with pleural plaques, "decrements when they occur are probably related to early subclinical fibrosis." Schwartz (1990), p.321, concurs. Similarly, Whitehouse (2004), p.224, states: "pleural changes alone are unlikely to cause a decrease in DLCO (diffusion capacity). DLCO decreases are likely to be associated with interstitial disease not apparent clinically on either plain chest radiograph or HRCT." Pleural plaques are associated with losses of lung function, and are probably associated with subclinical fibrosis.

26. It is universally accepted that pleural plaques are markers of asbestos exposure. ATS (2004) Official Statement, p.705 states: "the presence of pleural plaques should be interpreted as a marker for elevated

risk of malignancy." With or without pleural plaques, "it is clear that an excess risk for pulmonary carcinoma exists in asbestos-exposed workers in the absence of radiographic evidence of asbestosis." Fraser and Pare text, p.1075. The Rosenstock text, p.372, states: "plaques are also associated with increased risk for the malignant outcomes of asbestos exposure, including mesothelioma and lung cancer." Edge (1979) established that an asbestos exposure sufficient to cause pleural plaques is sufficient to cause mesothelioma.

27. An overwhelming majority in the Libby cohort have not only pleural plaques, but also diffuse pleural thickening, a more serious form of pleural disease. Serial observations of Libby miners over 30 years show many with pleural plaques showing on chest x-rays in the 1960s or 1970s, then development of diffuse pleural thickening in the 1980s, then increasing lung function loss in the 1990s, with death in the 1990s or after 2000.

28. Diffuse pleural thickening is pleural fibrosis, which is scarring from an inflammatory process due to the presence of asbestos fibers. ATS (2004) Official Statement, p.707 states: "Diffuse thickening of the visceral pleura is not sharply demarcated, and is often associated with fibrous strands "crowsfeet" extending into the parenchyma (lung structure) . . . Diffuse pleural fibrosis extends continuously over a portion of the visceral

pleura often causing adhesions to the parietal pleura."

The Fishman text, p.946, states: "Diffuse pleural fibrosis occurs most commonly as part of a fibrotic process of the visceral pleura and subadjacent interstitium. It may occur, however, and be quite severe in patients with minimal pulmonary parenchymal fibrosis." This is true in the Libby cohort as well. Light and Lee, Textbook of Pleural Diseases, p.501, states: "Diffuse pleural thickening appears more closely related to amphibole than chrysotile exposure."

29. Lee et al (2003), p.205, states with regard to 38 subjects with Wittenoom crocidolite exposure: "In our study, pleural thickening was associated with reduced lung volumes and DLCO (as had been shown by others 34, 35)." Yates (1996), p.304, states with regard to 64 patients with diffuse pleural thickening: "Our study confirms the findings of earlier workers in that a significant decrement of FEV_1 and FVC was observed (3-12, 15-22)." The ATS (2004) Official Statement, p.707, states: "Decrements associated with diffuse pleural thickening reflect pulmonary restriction as a result of adhesions of the parietal with the visceral pleura. Restrictive impairment is characteristic [meaning decreased vital capacity and decreased lung volumes]." The Rosenstock text, p.373, states: "In contrast to the mild effect of plaques on lung function, diffuse pleural

thickening may result in more significant restrictive respiratory impairment."

30. It is common knowledge that there is little correlation between radiographs and asbestos pleural disease severity, and certainly none that could be applied to individual patients. Miles et al (2008) "Clinical Consequences of Asbestos-related Diffuse Pleural Thickening, a Review," p.6, states: "Few longitudinal studies exist, but these have found no correlation between radiographic severity and longitudinal loss of lung function." Yates, et al (1996), "Asbestos re Bilateral Diffuse Pleural Thickening: Natural History of Radiographic and Lung Function Abnormalities," p.305, states: "No correlation was observed between radiographic severity and longitudinal loss of lung function. The chest radiograph even when accompanied by oblique films was an insensitive index of disease severity." Similarly, Whitehouse (2004), p.223, states: There was no statistical correlation between the extent of pleural changes measured on the chest x-ray and the loss of pulmonary function."

Similar statements have been presented in studies on the asbestos insulators. Markowitz et al (1997), p.106, states: "The chest x-ray can be normal even in the face of severe interstitial fibrosis." In the Markowitz et al (1997) study of 74 asbestosis deaths, 11% (8/74) had minimal or no interstitial fibrosis (ILO score of 0/1 or less). Selikoff et al (1964), p.148,

states: "significant disability may be present with relatively little to be seen on x-ray, and, conversely, x-ray changes may be extensive, with little functional difficulty."

E. CARD Mortality Study

31. A mortality study was done on patients of the Center for Asbestos Related Disease (CARD) Clinic in Libby. The cohort is all patients seen at CARD or by me in Spokane, with an asbestos-related disease diagnosis and chest films. The study was done under my supervision.

A total of 227 patients were identified as deceased through 7/9/08. 41 were excluded as having had no diagnosis of asbestos-related disease (ARD), no death certificate, no chart and/or no chest film, or as having had no exposure pre-1990. The total included in the mortality study for analysis is 186.

Data was gathered from patient charts. Available death certificates were obtained, including those available through the law firms of McGarvey, Heberling, Sullivan & McGarvey and Lewis, Slovak & Kovacich.

I reviewed patient charts and made determinations on whether asbestos-related disease was a significant contributing factor in the death of each patient, per the "best available information" method described in Selikoff et al (1992), "Use of Death Certificates in Epidemiological Studies,

Including Occupational Hazards: Variations in Discordance of Different Asbestos-Associated Diseases on Best Evidence Ascertainment." Data is displayed on the chart "Summary of Mortality Disease Percentages," (Exh. 7). I determined that 116 of 186 (62%) deceased patients died of asbestos-related disease - 7 from mesothelioma, 19 from asbestos related lung cancer, 11 from other asbestos related cancer, and 79 from "asbestosis" (including asbestos related pleural disease). Note that nonmalignant ARD deaths are generally coded to ICD-9 501 "Asbestosis."

Other important observations include the following.

1) Only 34% (39/116) of those who died of non-malignant disease were mineworkers. 66% (77/116) were community members and family members of mineworkers. Exh. 7.

2) The death rate from asbestos disease was not greatly different as between mineworkers (72% - 39/54) who generally had heavy exposures, and community members (54% - 58/108), who had light exposures.

3) 36% (27/74) of those who died of non-malignant disease died with pure pleural disease, with no interstitial fibrosis on the last chest x-ray. Of the 27, 8 had available CT scans. Of the 8, 3 had no interstitial disease on CT scan. Probably most of the 27 with no interstitial disease on chest x-ray did show some on CT scan. ATS (2004), p.707, reports only five total

cases of death by pleural disease outside Libby.

4) All 79 "asbestosis" deaths had pleural disease to some degree. One had no pleural disease on chest x-ray, but did have significant pleural thickening on CT scan. 36% (27/74) had no interstitial disease on chest x-ray. 46% (34/74) had minimal or no interstitial disease (0/1 or less), on chest x-ray. 75% (57/76) died with moderate or extensive pleural disease (50 with moderate or extensive pleural thickening and 7 with moderate or extensive pleural plaques). 26 of the 57, had minimal or no interstitial fibrosis (ILO 0/1 or less). This means that many had moderate or extensive pleural disease, and minimal or no interstitial disease. The above confirms clinical observations that many patients die with extensive pleural disease and little or no interstitial disease. We find no similar reports of a high death rate by asbestos pleural disease elsewhere in the literature.

5) 89% (68/76) had pleural thickening. 47% (36/76) had pleural plaquing. Note that pleural plaquing may not be seen where diffuse pleural thickening is extensive.

6) The last pulmonary function test on each patient was reviewed. Using severity designations appropriate to Libby patients, the following observations are made per Knudson norms for spirometry, Intermountain Thoracic Society for lung volumes and Miller for diffusion capacity. Use of

Intermountain norms for spirometry and diffusion capacity will result in a significant decline in percentage predicted:

2 normal (FVC, TLC, DLCO all over 79)
7 mild (FVC, TLC, or DLCO between 70 and 79)
12 moderate (FVC, TLC, or DLCO between 60 and 69)
55 severe (FVC, TLC, or DLCO under 60)
3 no PFT
79 total

7) It is significant that 44% (30/68) had only DLCO under 65, not FVC or TLC. DLCO is a very important indicator of severity in the Libby cohort.

8) 43% (32/75) had the FEV_1/FVC ratio under 65, indicating some obstructive defect. Only 8% (6/71) had an abnormal total lung capacity (over 120%), indicating that predominately obstructive disease was rare in the cohort.

9) Mean age at diagnosis was 69.4. Mean age at death was 76.3. Based upon male life expectancy at diagnosis, an average 7.5 years of life expectancy was lost to asbestos disease.

32. Comparison of CARD Mortality Study to Insulator Studies. The CARD mortality study found that in the group of patients diagnosed with ARD who have died, 48% died with ARD as a significant contributing factor per death certificate, and 62% died with ARD as a significant contributing factor per best available information analysis. Exhibit 7, Summary. The

62% death rate from asbestos disease appears to be the highest reported for any cohort in the United States.

The experience of the cohort of asbestos insulation workers provides a context for comparison of Libby mortality from asbestos related disease. The asbestos insulators had extremely heavy exposures to asbestos dust, often working in clouds of dust. The Libby patients, on the other hand, are mostly community members who had relatively light exposure to asbestos from casual exposure, such as breathing the air in the Libby area. Only 34% (39/116) of the Libby patients who died of asbestos disease were mine workers.

The cohort of asbestos insulation workers was first studied in Selikoff et al (1964). A number of follow-up studies have been performed, including Selikoff and Seidman (1991), "Asbestos Associated Deaths Among Insulation Workers in the U.S. and Canada, 1967-1987." The results of this mortality study and the CARD mortality study are compared in the following table.

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Selikoff and Seidman (1991), p.7, Table 2 summary format; comparison to Libby CARD mortality study

	Insulators per DC		CARD per DC		Insulators per BAI		CARD per BAI	
Lung cancer	1008	20%	16	9%	1168	24%	19	10%
pleural meso peritoneal meso	89 92	4%	7	4%	173 285	9%	7	4%
GI cancer	188	4%	9	5%	189	4%	11	6%
asbestosis	201	4%	58	31%	427	9%	79	42%
Total ARD deaths	1578	32%	90	48%	2242	45%	116	62%
All causes	4951	100%	186	100%	4951	100%	186	100%

DC - death certificate
BAI - best available information

One problem in comparing the two mortality studies is that in the CARD study all patients in the cohort were diagnosed with asbestos-related disease (ARD), whereas in Selikoff and Seidman (1991), there is no information on whether all the deceased had earlier been diagnosed with ARD. It can be estimated from Selikoff and Seidman (1991) that a very high percentage of the insulators who died likely had been diagnosed with ARD. From Table 4, we see that 96% (967/1,008) of the deaths from lung cancer were over 20 years from first exposure. From Table 6, we see that 96% (412/427) of the deaths from asbestosis were over 20 years from first exposure. From Selikoff et al (1964), Table 5, we can determine that 86% (339/392) of those workers over 20 years from first exposure had abnormal

chest x-rays. With the abnormal chest x-ray and exposure history, there is a likelihood of diagnosis of ARD. We conclude that the results displayed in the table above for the two studies are comparable. Even if we subtract 5% from the CARD death rate (since all CARD patients were diagnosed with ARD), the CARD death rate is still significantly higher than the insulators' death rate, despite the light exposure of the CARD patients.

The Libby death rate from ARD is 62% per best available information with 48% per death certificates. The insulators' death rate from ARD is 45% per best available information and 32% per death certificates. With a death rate higher than even the insulators cohort, it is apparent that exposure to Libby asbestos is considerably more toxic to humans than was the predominately chrysotile asbestos exposure of the insulation workers. (Regarding fiber type, see Seidman and Selikoff (1990), p.311).

Markowitz et al (1997), "Clinical Predictors of Mortality from Asbestosis in the North American Insulators Cohort, 1981 to 1991," followed 2,609 insulators examined in 1981 to 1983. The CARD mortality study may be compared to the Markowitz et al mortality study on asbestos insulators. The abstract states: "Seventy-four (11.0%) of 674 deaths during the subsequent 10 years were due to asbestosis, according to the best clinical and radiological evidence available at the time of death." The CARD mortality study used similar methods. Results are compared as follows:

	Markowitz et al (1997)	CARD Mortality Study
Mean age at examination	57.5	69.4
Mean age at death	65.9	76.0
Years exam to death	8.4	6.6
Asbestosis deaths	74	79
Asbestosis deaths as % of total deaths	11%	42%
No pleural abnormalities on CXR	11%	1%
No interstitial abnormalities on CXR	4%	37%
Interstitial 0/1 or less on CXR	11%	43%
Interstitial 2/1 or more on CXR	55%	12%

Though the studies differ in minor respects, they are epidemiologically comparable. Markowitz et al (1997) followed individual insulators for 10 years in sequence. In the CARD study, patients were followed from diagnosis to death, a period averaging 6.6 years, but in some cases was over 10 years. These studies are nevertheless comparable. Both studies used the designation "asbestosis," despite varying degrees of pleural and interstitial disease.

The insulators had predominately interstitial disease, whereas the CARD patients had predominately pleural disease. The insulators were about 4x as likely to have severe interstitial disease (55% to 12%). The CARD patients were about 4x as likely to have minimal interstitial disease (44% to 11%). Of the insulators group, 11% (8/74) were category zero for interstitial fibrosis. Of the eight in this group, seven had pleural thickening. Markowitz et al (1997), p.104. Of the CARD patients, 40% (30/74) were category zero for interstitial fibrosis. All but one of the 30 had pleural

disease on chest x-ray, and that one had both pleural and interstitial disease on CT scan.

Markowitz et al (1997) does not state the number who were diagnosed with asbestos-related disease on examination in 1981 to 1983. As discussed above, it is known that the incidence of asbestos-related disease was very high in the insulators with 30 years exposure. All patients in the CARD mortality study were diagnosed with asbestos-related disease.

The most striking observation is that the CARD patients' death rate from asbestosis is about 4x that of the insulators (42% to 11%). This result appears despite the important difference in exposure levels. 63% (50/79) of the CARD patients were family or community members with light exposures, whereas the insulators had industrial exposures to asbestos which were generally quite extreme.

It is also observed that the CARD patients once diagnosed have a probability of death by asbestos-related disease, whereas the insulators do not. Markowitz et al (1997), Table 2, shows that no category (except ILO 3/4 which is exceedingly rare) of interstitial or pleural disease brings a probability of death.

F. Libby asbestos is highly toxic.

33. Many studies have been done on Libby asbestos and its toxicity. See the attached listing: "Libby Studies."

34. There generally appears to be a distinct pattern for Libby asbestos disease. The disease appears to be predominately pleural, for the large portion of the time that people have the disease. In the Whitehouse (2004) study, 55% of the patients followed had no interstitial disease and 45% had only minimal interstitial disease. The percentage of patients in the overall patient cohort with no significant interstitial disease is even higher now, as more recent diagnoses include many people in the early course of the disease. By contrast, lung function loss in asbestos disease from predominately chrysotile exposures is mainly secondary to interstitial disease. In the Libby cohort, interstitial disease generally becomes radiographically visible rather late in the process, and frequently is only a minor factor.

Consistent with the pleural pattern, in the CARD mortality study 46% (34/74) died with minimal or no interstitial disease (1/0 or less). There was more interstitial disease in the CARD mortality study than in the 123 patients studied in Whitehouse (2004). This is consistent with my clinical observation that interstitial disease often appears in the last years before death.

Consistent with the pleural pattern is the statement in the Peipins et al (2003) abstract: "We observe pleural abnormalities in 17.8% of participants and interstitial abnormalities in less than 1% of participants undergoing

chest radiography."

35. Pleural disease in the Libby area is widespread. The patient population is not concentrated in small geographical areas of higher intensity dust contamination from W.R. Grace mining activities. Peipins et al (2003), abstract, states: "Mining, handling, processing and personal or commercial use of asbestos-contaminated vermiculite have led to widespread contamination of the Libby, Montana area." Peipins et al (2003), p.1754, states: "air sampling in downtown Libby in 1975 and at several points in the 1980s detected levels of asbestos well above the OSHA occupational limit of 0.1 fiber/cm3 over eight hours of exposure." A W.R. Grace report, Source Emissions, Results of Surveys, 1975 is attached as Exh. 20. Asbestos fiber levels were stated as 0.67, 1.5 and 1.1 for three locations in the town of Libby in 1975. The town is about seven miles from the mine.

As to the population of the Libby area, Peipins et al (2003), p.1758, speaks of a population of "9521 persons in Central Lincoln County, a population that has been relatively stable for the past 30 years (U.S. Bureau of the Census 2002)." Peipins et al (2003) discusses the demographics of study participants and discusses the results of screenings administered by the ATSDR in 2000 and 2001. Chest x-rays were taken on 6,668 participants, representing 61% of the area population, of whom 18% (1,186 of 6,668) had pleural abnormalities. This is a very large number for a small

community.

Peipins et al (2003), Table 3, notes the exposure pathways for the 1,186 with pleural and/or interstitial abnormalities:

Ever worked for W.R. Grace	186
Lived with W.R. Grace workers	358
Environmental only exposures derived as 1186-358-186 =	642
Resident (more than 6 months before 1991)	1,186.

Note that all participants are "residents." Peipins et al (2003), p.1755, states "the pathways presented here are not mutually exclusive." Many community exposure pathways are listed. Most study participants with abnormalities are not workers or family members of workers, but are simply residents of the Libby area.

The pattern seen in exposure histories for patients of the CARD Clinic is consistent with the findings in Peipins et al (2003). Most patients are community members who are not workers or family members of workers. Indeed the percentage of new patients who are community members has increased in recent years. An important aspect of Libby area exposures is that residents had a 24 hour exposure, with no respite away from exposure to clear out the airways. This appears to be generally different from exposures reported elsewhere.

The CARD Clinic has diagnosed over 1,800 patients with asbestos related disease by either plain chest x-ray or CT scan, and has confirmed diagnoses on about 100 patients in other areas of the U.S. All had exposures to Libby asbestos due to W.R. Grace or Zonolite Company operations in or near Libby, Montana. Very few patients had exposures to other asbestos outside the Libby area. Few patients had non-vermiculite related asbestos exposures within the Libby area. All patients in the over 1,800 diagnosed above have asbestos disease due to exposure to Libby asbestos with its source in W.R. Grace and Zonolite Co. mining and other activities.

36. The following numbers are supplied by the McGarvey, Heberling, Sullivan & McGarvey law firm. Of 653 Libby Claimants who are patients of CARD:

Ever worked for W.R. Grace	189	29%
Subcontractors at W.R. Grace	26	4%
Family household member of W. R. Grace worker	143	22%
Community members (all others)	295	45%
Total	653	100%

See printout, Exh. 4 "Client Sort by Exposure (Community, Family Member, Worker)" (CARD patients only) 5/28/08. While these numbers are representative only of Libby Claimants, they are consistent with the findings in Peipins et al (2003), in that the greatest number are community members,

who typically played baseball near Grace operations, played as children on piles of Grace vermiculite, or as residents breathed in asbestos in the dust in the air in the Libby area.

37. In addition to the vermiculite contamination widespread in the Libby area, a contributing factor in the widespread pleural disease is the minimal exposures which triggered disease. There are many examples in a patient cohort of surprisingly minimal exposures which led to significant disease:

> Tom Murray, a former federal magistrate judge, worked at the mine during the summers of 1948 and 1949, and died of asbestos lung cancer.
>
> Betty Maxwell who only visited Libby for 1-2 week periods for about ten summers, and cleaned cabins, now has severe asbestos disease and is on oxygen.
>
> Victoria Skidmore who only visited Libby for errands averaging about two trips per month for 14 years, now has pleural mesothelioma.

38. A hallmark of pleural disease from Libby asbestos exposure is that it is highly progressive. In Whitehouse (2004) (Exh. 2), 123 patients with two or more sets of pulmonary function tests were studied. All had pleural disease, and 55% had no evidence of interstitial changes. There was an average of 35 months between first and last lung function test. 76% of the patients showed progressive loss of pulmonary function. The extent and rapidity of loss of pulmonary function were highly significant. Page 221

states that for the 123 patients "the average yearly loss was 2.2% for FVC, 2.3% for TLC, and 3.0% for DLCO." To my knowledge, no study of asbestos disease from predominately chrysotile exposure shows overall progression rates for lung function loss anything like those seen in Libby, where most patients have had relatively light exposures.

As of 8/23/07, 28 of the 123 patients in the study had died. For 25 of 28, asbestos related disease was a significant factor in death. Life expectancy at date of diagnosis was calculated for each of the 25. The average loss was 9.5 years of life expectancy for the 25 patients. See Exh. 5. "Dead in 123 patients in Whitehouse (2004)."

39. Interestingly, Alfonso (2005), an amphibole study from Australia, similarly finds an "average rate of decline of DLCO . . . [of] about 2.2% per year [0.55/24.8] in people with asbestosis." Asbestosis was considered present upon an ILO score of 1/0 or greater. DLCO decline was not investigated for pleural disease in the study cohort. In another amphibole study, Cookson (1983), abstract, states "[t]he ratio of transfer factor to effective alveolar volume correlated directly with the degree of pleural thickening as alveolar volume fell with increasing severity of pleural disease."

Sichleditis (2007) is a study of environmental exposure to tremolite asbestos in Almopia, Greece. It finds significant progressive lung function loss in patients with pleural disease. Villagers whitewashed buildings using

mineral dust, which contained tremolite asbestos. A doubling of the size of pleural plaques was found in 126 subjects followed for 15 years. 18 of the 126 had pulmonary function tests in 1988 and 2003. Percent predicted for total lung capacity dropped from 96% to 76% (1.3% per year), forced vital capacity dropped from 95% to 80% (1.0% per year), and residual volume dropped from 98% to 66% (2.1% per year).

The 18 subjects with pulmonary function tests had comparatively mild radiographic progression, with only a 60% increase in area of pleural plaques (p.636) (data at Table 2, p.640). The authors report that "no obvious radiological abnormalities were detected in lung parenchyma," p.636.

40. Rapid progression in the Libby cohort can also be seen radiographically. Exhibit 6 is a Chart titled "Chart Case # Type of Progression," which presents a collection of 18 cases of rapid progression in seven years or less, secondary to pleural disease and/or interstitial disease. To my knowledge, progression of pleural disease of this nature has not been reported elsewhere.

41. Pleural disease from exposure to Libby asbestos appears to be far more severe than asbestos pleural disease reported elsewhere. The CARD mortality study presents 27 deaths by pleural disease with no interstitial disease on last chest x-ray. Exh. 7. ATS (2004) p.707, reports only five total cases of death by pleural disease outside Libby. I have read

reports of others, but they remain very few. In Miles et al (2008), p. 2, the Australian authors report that death secondary to diffuse pleural thickening is "very rare." David Austern, Administrator of the Johns-Manville Trust, reports that in the history of the trust there has been just one death by asbestos pleural disease. (D. Austern, personal communication). Commonly the mechanism for death by pleural disease is loss of pleural space resulting in episodes of hypoxia. Due to the highly progressive nature of Libby asbestos disease, once diagnosed with pleural disease, with multiple pleural plaques or diffuse pleural thickening and a loss of lung function, a patient has a high probability of progressive disease and early mortality due to asbestos disease.

42. The CARD mortality study observed that 65% (117/186) of deceased patients of CARD died of asbestos disease, meaning that asbestos related disease was at least a significant contributing factor in the death. See Exh. 7, Summary of Mortality Study Disease Percentages. Cause of death per death certificate was noted, and also a determination was made by best available information review of medical records, per Selikoff (1992). In contrast, a patient diagnosed with asbestos disease from predominately chrysotile exposure has a much lower likelihood of death.

43. In the CARD pleural disease cohort, there is a large number of patients with severe pulmonary function impairment. To my knowledge, no

similar incidence of impairment is reported in an asbestos pleural disease cohort elsewhere, or in a chrysotile disease cohort elsewhere. Note that some Libby patients have severe impairment mainly due to smoking. A proper cohort comparison would include cohorts with asbestos pleural disease or chrysotile disease including all smokers.

Per the records of the McGarvey, Heberling, Sullivan & McGarvey Law Firm, of 653 Libby clients with asbestos disease from exposure to Libby asbestos who are also patients of CARD, 77 are on oxygen, and 171 have at least one of the three main indicators of severity of asbestos disease under 60% of normal (FVC, TLC or DLCO). Exhibit 8. While these numbers are representative of the listed patients only, based on clinical observation, it is likely that they are indicative of severity of asbestos disease in the CARD patient cohort. (Miller norms are generally used for DLCO. If translated to Crapo, a decrease of roughly 10% of predicted occurs.)

A number of studies report an increased incidence of extensive fibrosis and pleural effusions associated with exposure to Libby asbestos, as compared to what is seen in other populations. See Lockey (1984), McDonald (1986), Amandus (1987) and Libby Studies listed.

44. Attached is a chart titled "Mesothelioma Cases with Exposure to Libby Asbestos as a significant factor." (7/23/08). Exhibit 9 (and CD with data). 31 cases of mesothelioma are verified, where exposure to Libby

asbestos was a significant factor in producing the mesothelioma. Three others await verification. Verification is per death certificate or pathology report, or both. This is an extremely high incidence of mesothelioma in a community with an average population of 9,521. Peipins et al (2003); ATSDR (2002). The background mesothelioma rate is thought to be about one per million per year. Roggli (2007). There have been 20 mesotheliomas with exposure to Libby asbestos as a significant contributing factor in the past 12 years (1996-2007), or 1.7 per year. If one uses the 20 mesotheliomas, then the Libby area, with a population of about 10,000 is at 166 per million per year, or 166x the background rate. If one uses the 10 mesotheliomas with the Libby area as residence at death, the rate is 83 per million per year, or 83x the background rate. Libby's mesothelioma rate is certainly the highest in the United States. The Fraser and Pare text, p.886, states the mesothelioma rate to be "33 per million per year for South Africa and to be 66 per million per year for Western Australia." Libby's mesothelioma rate is among the highest in the world. It is also noted that the crocidolite mine at Wittenoom, Australia closed in 1966, whereas the Libby mine closed in 1990. The full manifestation of mesothelioma in Libby is far from completion.

45. Libby has also had 13 environmental exposure mesothelioma cases (1995-2007) where the exposure to Libby asbestos is considered to

be a significant factor in producing the mesothelioma. Exh. 9. 11 cases were described in Whitehouse et al (2008). Two more have been verified since the date of submission of the article. "Environmental" excludes miners, but includes immediate family members of miners. Two mesothelioma cases are household family members of workers at the mine (Orem and Flatt). If one uses 11 environmental cases 1995 -2007, this could translate to 0.85 per year (11 cases in 13 years), or 85 Libby environmental mesothelioma cases per million per year. However, one is alive and only five of the 11 died as Libby residents. Many Libby patients in end stage disease move to areas with major medical centers. If we use only the five who died as Libby area residents, then the rate is 38 per year, or 38 per million per year. Libby's environmental mesothelioma rate is the highest in the United States, and is among the highest in the world.

Berry (1996), "Mesothelioma Incidence and Community Asbestos Exposure," presents mesothelioma statistics for workers and community residents near the Johns-Manville Plant in the City of Manville, Somerset County, New Jersey. The Manville plant operated 1912-1980. The Libby mine and mill operated from the 1930s to 1993. Even though the Libby mine operated for fewer years and closed 13 years later, such that the fulminate presentation of mesothelioma cases is yet to occur, the Libby community mesothelioma rate appears to be higher than that of Somerset

County, New Jersey.

The Manville plant "employed up to 3,500 people," p.34. The Libby mine and mill employed at most about 160 people, a difference of about 22x. The town of Libby has had a population of about 3,000. The town of Manville, situated adjacent to the plant, had an average population of 10,923, p.36. Central Lincoln County (defined as a 10 mile radius from the center of Libby) had 9,541 persons, Peipins (2003). Somerset County, New Jersey, had an average population (1980 and 1990) of 208,435. This is about 22x the size of Central Lincoln County. So, whereas the Manville workforce was about 22x of that of Libby, the community size from which mesothelioma cases is drawn is also about 22x that of Libby.

Berry (1996), shows total mesothelioma cases for 1979-1990 for Somerset County at 143. 61 plant employee cases are subtracted out, for a net community case number of 82. Using 82 community mesothelioma cases in 208,435 residents over 11 years results in 36 per million per year. Using 11 Libby community mesothelioma cases in 9,541 residents over 11 years (1995-2006), results in a rate of 105 per million per year.

Berry (1996), p.36, used "address at time of diagnosis" to determine eligibility. If we apply this to the Libby community mesothelioma cases, then we find five with Central Lincoln County residence at the time of diagnosis.

Using these five cases results in a mesothelioma rate of 48 per million per year (5/11 x 105), exceeding Somerset County, New Jersey at 36 per million per year.

Berry (1996), p.38, states that particulate from the Manville plant "regularly coated cars, homes and yards like a fresh snowfall in the immediate community." The Libby mine and mill were seven miles northeast of Libby, with a prevailing wind from the west and southwest. The entire production of the mine was shipped out of the Libby railroad yard in town, and W.R. Grace had a small expansion plant and bagging plant near the railroad tracks. Libby residents report substantial dust in the town, but it appears that the Manville asbestos dust concentrations in town were significantly greater. However, the Libby asbestos is amphibole asbestos, whereas Manville used approximately 95% chrysotile asbestos, p.38. Amphibole asbestos is more toxic, which likely accounts for the greater rate of mesothelioma cases in the Libby community, even though exposures were not as heavy.

Berry (1996), notes that due to insufficient exposure histories, there could be no sorting of community (non-worker) cases into household member of worker cases and pure environmental cases. This sort can be accomplished with the Libby community mesothelioma cases.

46. Sullivan (2007), Table 1, conservatively finds as of 2001, 154 deaths among Libby mineworkers from asbestos related disease (99 lung cancers, 15 mesotheliomas and 40 asbestosis). Sullivan (2007), Table 2, also finds 111 miner deaths by nonmalignant respiratory disease (NMRD). This is a larger catchall category, and includes the 40 asbestosis deaths. In all likelihood, if the Selikoff (1991) best available information analysis were applied, many of the other 71 NMRD deaths in Libby miners could be identified as due to asbestos related disease. The Sullivan (2007) count of 154 miner deaths due to asbestos disease is very conservative.

47. The CARD mortality study adds another 27 miners (including subcontractors) who have died from asbestos related disease since 2001. Exh. 7 in addition to the subjects in the mortality study. 23 more workers' (including two subcontractors) death certificates and records have been reviewed by me (Exh. 10). These 23 ARD deaths were persons not seen at CARD or by me in Spokane. The total for workers is 204 (154 + 27 + 23).

The CARD mortality study shows 50 family members or community residents died of ARD. In addition to the subjects in the mortality study, 14 more family and community members' death certificates and records have been reviewed. Exh. 10. These 14 ARD deaths were persons not seen at CARD or by me in Spokane. The total for family and community members is

62 (48 + 14). The above conservatively totals 264 (204 + 62) deaths in the Libby cohort due to asbestos-related disease.

Studies on deaths in the Libby cohort are summarized on the attached chart "Libby Cohort Deaths per Source," Exh. 21. It also appears that W.R. Grace had documented 30 lung cancers as of 1985. See Exh. 22, memo of 9/17/85.

48. "Lincoln County, Montana, had the highest age adjusted asbestosis mortality rate in the United States for 1988-1997. (Castellan R. unpublished data)," letter by Peipins et al in Environmental Health Perspectives 112:a83. It appears that this statement applies to 1998-2007 as well.

ATSDR (2002), "Mortality in Libby, Montana 1979-1998," is a death certificates study. Some results from ATSDR (2002) were published in Horton et al (2006), "A Review of the Federal Government's Health Activities in Response to Asbestos-Contaminated Ore found in Libby, Montana." ATSDR (2002), p. 1, states the following conclusions:

> For the 20 year period reviewed in this report (1979-1998), mortality in Libby resulting from asbestosis was 40 to 80 times higher than expected. Mortality from lung cancer was also elevated, with a 20 to 30 percent excess over this time period.

Table 10 shows 12 deaths in category 501 "asbestosis," resulting in standardized mortality ratio 65x higher than that of the U.S. reference

population.

ATSDR (2002) is a very conservative study, since it apparently used only the primary cause of death, not underlying or contributing causes, and only counted those who were Libby residents at death. Libby's end stage patients often move to larger towns with major medical centers, and die there. ATSDR (2002) may have understated the incidence of asbestosis deaths in the Libby area. It is not clear how the U.S. reference population incidence of asbestosis was calculated, and whether only the primary cause of death was used for the U.S. reference population.

We will apply the ATSDR (2002) approach of using only the death certificates' primary cause of death, to the data in the CARD mortality study. We also include only those who died as residents of the "Libby area," defined as "Central Lincoln County," per ATSDR (2002). For the ten years 1998-2007, the result is 10 for category 501 "asbestosis." Exh. 7. We double the ten year total for comparison of the ten year rate to the ATSDR (2002) 20 year U.S. baseline rate. This assumes that the baseline U.S. death rate for asbestosis has not significantly changed from that used in ATSDR (2002). (1998-2007: 10 asbestosis deaths x 2 = 20. 65 x 20/12 = 104). It appears that the death rate for 1998-2007 is 104x the U.S. rate. The Libby area has the highest asbestosis mortality in the United States.

49. There is a latency period between exposure and the first

appearance of asbestos disease on chest x-ray or CT. During the latency period, microscopic asbestos fibers are working at a microscopic level, until they become detectible on chest x-ray or CT. ATS (2004), p.695, suggests a minimum latency period of 15 years. Asbestos pleural disease from exposure to Libby asbestos often appears radiographically in less than what would be considered a minimum latency period of 10-15 years. We find no report of short latency in disease from predominately chrysotile exposures.

Data on Libby asbestos disease indicates that changes of asbestos disease can occur in as little as 3-5 years (clearly defined plaques), whereas chrysotile latency periods are generally over 10 years. With Libby asbestos, the range generally appears to be about 5-40 years, with an average latency period of about 15-30 years from first exposure to diagnosis. Miles et al (2008), p. 3, report that "DPT can develop within a year from exposure to asbestos." We have not seen a latency that short in cases of Libby exposure.

The results of W.R. Grace's annual chest x-ray program for workers from 1959 to 1980 appear to confirm that the Libby asbestos has a very high toxicity. Grace's in-house studies in 1969, 1975 and 1976 on annual x-rays showed lung abnormalities in up to 17%, 8% and 6% of employees with under six years of work. (Exhibit 11, Charts 035a, 035b and 035c attached). While these workers likely had community exposures prior to

going to work for W.R. Grace, it is also likely that exposure at Grace was their first high intensity exposure.

Consistent with these findings is Cookson (1986), Fig. 1, finding that about 20% of workers with abnormal films reached "onset of asbestosis (i.e. progression from category 0 to category 1)" (p.996) after just 8 years of work exposure to amphibole asbestos (Exhibit 12).

Disease at an early age is common in the Libby cohort. Per CARD Clinic records, 65 patients are in their 30s and 189 patients are in their 40s. This statement is based upon a total of 1,957 patients on 9/13/06.

50. DLCO (diffusion capacity) is a particularly important indicator of the severity of impairment in the Libby asbestos disease patients. We find that the DLCO defect is the leading indicator of severity in the Libby cohort, and has the greatest correlation with shortness of breath and the timing of the patient's entry to oxygen treatment. In some cases there is significant asbestos disease on the chest x-ray and only the DLCO is reduced, not the FVC or TLC. Some patients with severe shortness of breath are severe only in the DLCO defect. In Whitehouse (2004), p.224, 76% of the 123 patients had progressive loss of lung function. Losses were about the same for FVC, TLC and DLCO at 2%-3% per year.

In the CARD mortality study, among those who died of nonmalignant asbestos diseases, 44% (30/68) had only DLCO under 65 (out of FVC, TLC

and DLCO).

The Rosenstock text, p.371, states: "The earliest and most sensitive finding in asbestosis is frequently a diminished diffusion capacity, which may occur in isolation or in combination with other findings." The 2005 Public Citizen comment by Dr. Michael Harbut, Dr. Philip J. Landrigan, Dr. Alan C. Whitehouse and Dr. L. Christine Oliver states that DLCO is "essential to determine how badly a person's lungs are impaired." Exh. 16.

DLCO (diffusion capacity) defect is probably associated with subpleural interstitial fibrosis. Whitehouse (2004) explains:

> Pleural changes alone are unlikely to cause a decrease in DLCO. DLCO decreases are likely to be associated with interstitial disease not apparent clinically on either plain chest radiograph or HRCT.

It does not appear that decline in diffusion capacity (DLCO) is significant in U.S. cohorts with predominately chrysotile exposure.

51. Similarly, Alfonso (2005), in an amphibole study, finds an "average rate of decline of DLCO . . . [of] about 2.2% per year [0.55/24.8] in subjects with asbestosis." Alfonso (2005), p.184, also states: "Compared with never smokers, current smokers and ex-smokers had lower DLCO at baseline, but smoking status did not affect the change in DLCO during the follow-up period (four years for men)." Wang (1996), Figure 3 shows that asbestos exposure depresses DLCO more than does smoking.

In another amphibole study, Cookson (1983), abstract, states "[t]he ratio of transfer factor to effective alveolar volume correlated directly with the degree of pleural thickening as alveolar volume fell with increasing severity of pleural disease." Cookson, p.660, observes that reduced DLCO "in patients with asbestos induced pleural disease suggests covert parenchymal asbestosis." This is consistent with the statement from Whitehouse (2004) quoted above.

52. Chronic severe pleural pain occurs in a significant percentage of the Libby cohort at one time or another in the course of the disease. It is common in Libby patients with diffuse pleural thickening. It is often initially misunderstood by outside physicians as cardiac chest pain. Lockey (1984) similarly reports pleural pain in a cohort of workers in Ohio who were processing Libby vermiculite. Mukherjee et al (2000) report on a group of mineworkers and residents of Wittenoom, Australia, all exposed to amphibole asbestos (crocidolite). 43% (556 of 1,280) report chest pain (abstract). Yates (1996), p.304, in another amphibole study reports "chest pain is a common feature of DPT." For non amphibole exposures, "chronic severe pleural pain is rare in patients with asbestos related pleural disease." ATS (2004), p.702. The Fishman text, p.946 states regarding diffuse pleural thickening: "The diffuse nature of the lesion often leads to pulmonary symptoms, including dyspnea on exertion, chronic dry cough, and

chest pain." ATS (2004) Official Statement, p.695, states that generally in asbestos disease: "a non-productive cough is commonly present." A chronic dry cough is common in Libby patients.

G. Amphibole toxicity.

53. Amphibole asbestos in general and Libby asbestos in particular are more carcinogenic and fibrogenic (productive of asbestos related disease) than is chrysotile asbestos. The Greenberg text, p.480, states:

> Several studies have also shown that worker cohorts exposed to higher concentrations of amphibole fibers have higher lung cancer rates than those exposed to similar concentrations of chrysotile asbestos. . . . This pattern of increased toxicity of amphiboles also holds true for all the other asbestos-related lung diseases (asbestosis, pleural disease, and mesothelioma).

ATS (2004) Official Statement, p.693 states that a reason given for the greater toxicity of amphiboles is that chrysotile fibers "are cleared more efficiently than amphibole asbestos fibers, which may be retained indefinitely." ATS (2004) Official Statement, p.693. See also McDonald (2004), p.366.

54. The Fraser and Pare text, p.1075, states "exposure to amphibole fibers . . . is associated with a significantly greater risk of carcinoma compared to chrysotile exposure."

55. Amphibole asbestos appears to be at least 4x as carcinogenic as

chrysotile. Hodgson et al (2000) (abstract: the "risk differential between chrysotile and the two amphibole fibers for lung cancer is thus between 1:10 and 1:50"). Antman (1993), p.373S, states: "amphiboles are about 10 times as carcinogenic as chrysotile." EPA (2003) Final Draft, p.1.4 states that: amphiboles are about 4x as carcinogenic as chrysotile; EPA (2003) Report, p.3-1 states: "according to the proposed risk assessment methodology, amphibole fibers have a five fold greater lung cancer potency than do chrysotile fibers." Also, see Stayner (1996), abstract: "there is little evidence to indicate lower lung cancer risk" for chrysotile.

56. Tremolite asbestos, which appears to be a close relative to the Libby amphibole asbestos, is considerably more carcinogenic than chrysotile asbestos. See McDonald (1997), Table 1. American Thoracic Society (1990), p.1456, states: "[a]sbestiform varieties of tremolite, are highly carcinogenic." Case (1991), p.494, states regarding an animal study: "[s]ignificantly, the tremolite fibers were amongst the most carcinogenic tested, with actual incidence of 75% and 'percent tumor probability' of 100%." Also, McDonald (2004), calling the Libby asbestos "fibrous tremolite," found in a cohort of 406 Libby miners a 240% increase in respiratory cancer (Table 2).

57. Amphibole asbestos fibers are variously estimated at 100x to

1,000x as productive of mesothelioma as chrysotile fibers. EPA (2003) Final Draft, p.1-4 uses a factor of 1,000x ("for mesothelioma the best estimate of the coefficient (potency) for chrysotile is only 0.0013 times that for amphibole"). Hodgson (2000), abstract, uses a factor of 100x to 500x, ("at exposure levels seen in occupational cohorts, it is concluded that the exposure specific risk of mesothelioma from the three principal commercial asbestos types is broadly in the ratio of 1:100:500 for chrysotile, amosite and crocidolite respectively").

58. Amphibole fibers are more fibrogenic than are chrysotile fibers. McDonald (1999) (abstract: "this study suggests that amphibole fibers, including tremolite, are more fibrogenic than chrysotile, perhaps to the same extent that they are carcinogenic"). The same study indicates at Table 1 that Quebec tremolite asbestos fibers are at least two times as fibrogenic as chrysotile. McDonald (2004) demonstrates that in 406 Libby miners, deaths due to nonmalignant respiratory disease were at 309% of the U.S. rate.

59. Amphibole asbestos is more than twice as likely to produce asbestosis and asbestos pleural disease which is radiographically progressive, than is chrysotile asbestos. Compare the following predominately chrysotile studies: Jones (1989), Gregor (1979) and Becklake (1979), with the following amphibole studies: Sluis-Cremer (1989), Cookson

(1986), Ehrlich (1992), and McDonald (1999). See the chart "Studies on Radiographic Progression of Asbestos Disease." Exhibit 13.

In most patients with asbestos disease (including asbestos pleural disease) from exposure to amphibole asbestos, the asbestos disease is progressive. Sluis-Cremer (1989), p.852, states regarding subjects with predominately amphibole exposure, "it appears that once a dose of asbestos sufficient to initiate the disease has been retained, it is inexorably progressive." Mossman and Churg (1998), p.1669 state: "Biopersistence is probably responsible for the much greater tendency of amosite or crocidolite-induced asbestosis to progress, compared to chrysotile-induced asbestosis."

Cookson (1986), presents Fig. I, (attached as Exhibit 12) a chart showing that 34 years after first crocidolite exposure approximately 97% of workers with abnormal films progressed radiographically to at least mild disease, 77% to at least moderate disease and 65% to at least severe disease. Crocidolite, like Libby asbestos is an amphibole. Based on my experience, I believe the numbers for the Libby workers would be similar. Cookson (1986) is consistent with the high rate of functional disease progression (76%) found in Whitehouse (2004) in 123 patients with exposures to Libby amphibole.

H. Obstructive Defect and asbestos-related disease.

60. Restrictive disease restricts what is breathed in. Obstructive disease obstructs what is breathed out.

Asbestos disease has been generally thought to be predominately a restrictive disease. The scarring in the lung lining (pleura) and in the lung air sacs and structure (parenchyma) restricts the lungs' ability to expand on inhalation.

Smoking disease is an obstructive disease. It obstructs what is breathed out. With emphysema, the lung tissue acts like an over expanded balloon. It does not constrict back to its natural form. Hence exhalation is obstructed.

61. Smoking causes emphysema and chronic bronchitis. ATS (1995), p. 578, states:

> **Emphysema** is defined as abnormal permanent enlargement of the air spaces distal to the terminal bronchioles accompanied by destruction of their walls and without obvious fibrosis.
>
> **Chronic bronchitis** is defined as the presence of chronic productive cough for three months for at least two successive years.
>
> **Chronic obstructive pulmonary disease** (COPD) is defined as a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema.

ATS (1995), p.79, states: "[o]nly about 15% of cigarette smokers develop clinically significant COPD."

62. Generally the differences between obstructive disease due to smoking and restrictive disease due to asbestos can be sorted out on pulmonary function tests. The sorting process is seriously complicated by the fact that asbestos disease often causes airway obstruction, or obstructive disease.

63. It is generally accepted that ARD causes obstructive disease. The ATS (2004) Official Statement, p.710, states: "The association between airway obstruction and exposure to asbestos has been well demonstrated in non-smokers, and in some studies the association between exposure and airway obstruction is seen only among non-smokers." And, ATS (2004) Official Statement, p.708, states "asbestos exposure has long been known to be associated with an obstructive physiological abnormality." The Fishman text, p.950, states: "mild airway obstruction can also be seen in non-smokers with asbestosis." The Rosenstock text, p.371, states: "In addition to parenchymal effects, asbestos can cause air flow obstruction." The Fraser and Pare text, p.2445, states: "Many patients, however, show some degree of airway obstruction as well, as a result of asbestos-induced bronchiolar fibrosis." This means association with obstructive defect.

More recently Ohar et al (2004) reviewed data on 3,383 asbestos exposed workers in "the Selikoff registry." At page 751, the authors state:

"In this analysis of 3,383 subjects, we have shown that airways obstruction is more common than restriction in asbestos exposed individuals currently undergoing evaluation. These results confirm the findings of Kilburn and colleagues (31, 39, 40, 43) and supports published clinical pathologic correlates. Churg et al (46) have shown that asbestos exposure results in small airways disease. . . . in this study the effects of exposure to both cigarette and asbestos acted additively to induce airways obstruction."

64. The Fishman text, p.604-605, states: "the hallmark of the obstructive pattern is a reduction in the FEV_1/FVC percentage . . . Typically, all three lung volumes - residual volume, functional residual capacity, and total lung capacity are increased." Normal for FEV_1/FVC is generally 70 percent or higher. Markowitz, et al (1997), p. 102, used a ratio of 70 as normal for ages under 60 and a normal of 65 for those 60 or older. I concur with that. For hyperinflation in obstructive disease, TLC or RV must be abnormal, at over 120 percent of predicted.

65. A restrictive pattern is generally described as a reduction in forced vital capacity (FVC) and total lung capacity (TLC), with the FEV_1/FVC ratio remaining normal. The Fishman text, p.607, states: "The diagnosis of restriction is based upon the finding of a normal FEV_1/VC (ratio) and reduced VC in the setting of a reduced TLC . . . residual volume is usually reduced."

66. Very often there is an obstructive pattern associated with Libby amphibole asbestos disease. An obstructive pattern has been associated with asbestos pleural disease in the literature. ATS (2004) Official Statement, p.708 states: "Asbestos related chronic airway obstruction may result in reduction in the FEV_1/FVC ratio, associated with reduced FEV_1 (29, 76, 113, 127)." Schwartz (1990), p.323, states "diffuse pleural thickening was associated with modest decrements in the FEV_1/FVC ratio." See also Schwartz (1990), p.323, Table 4. Obstructive defect, with a reduced FEV_1/FVC ratio, is a significant factor in many Libby patients with diffuse pleural thickening, and may become severe enough to cause significant obstructive airway disease in the absence of any other cause. As noted by Ohar et al (2004), where the asbestos exposed patient also has a history of smoking, the two may be additive in causing obstructive defect.

67. ATS (2004) Official Statement, p.697, states: "Mixed restrictive and obstructive impairment is frequently seen; isolated obstructive impairment is unusual." The Rosenstock text, p.371 states: "A mixed restrictive and obstructive impairment is also common (with greater reductions in FEV_1 than FVC, resulting in a diminished FEV_1/FVC ratio. . . . Asbestos-related airflow obstruction interacts with the effects of smoking, resulting in more pronounced obstructive changes. . . . Total lung capacity

(TLC) is generally an insensitive measure of functional impairment. A result of the competing forces on TLC in mixed restrictive and obstructive impairment, with a reduction in TLC related to restrictive disease and an increase in TLC related to concurrent obstructive air trapping." ATS (2004) Official Statement, adds "total lung capacity may be normal when both disorders are present, due to a restrictive process offsetting air trapping." Likewise "the obstructive - restrictive combination can produce an overall normal spirometric test result." 2005 Public Citizen comment by Dr. Michael Harbut, Dr. Philip J. Landrigan, Dr. Alan C. Whitehouse and Dr. L. Christine Oliver. Exh. 16.

Mixed disease is frequently seen in the Libby cohort. Interestingly, in the Markowitz (1997) study of asbestos insulators at Table 5, mixed restrictive and obstructive disease produced a higher risk of death than did restrictive disease alone or obstructive disease alone.

I. Other studies on Libby patients.

68. In 2000, I performed a review of causes of death in certain workers at the W.R. Grace mine. This was not a comprehensive study. Available death certificates and medical records were reviewed. Most of the death certificates came from the records of W.R. Grace & Co. I identified 100 workers from the W.R. Grace mine and mill who had died of asbestos

related disease. Of the 100, 49 died of asbestos lung cancer, 11 died of mesothelioma and 40 died of asbestos related fibrotic disease (including asbestos pleural disease). See Exhibit 14 "Workers Dead from Asbestos Disease."

McDonald (2004) followed up on a cohort of 406 Libby miners. That study found, as of 1998, 107 total deaths, with 44 of respiratory cancer, 12 of mesothelioma and 51 of nonmalignant respiratory disease (NMRD, a somewhat broader category than asbestos related disease). The numbers in McDonald (2004) and the evaluation I did appear to be quite similar.

Sullivan (2007) followed up on the entire cohort of 1,672 Libby mineworkers. Sullivan (2007), Table 1, finds as of 2001, 767 total deaths, with 99 by lung cancer, 15 by mesothelioma, 40 by asbestosis and 111 by nonmalignant respiratory disease NMRD (which include the 40 asbestosis deaths).

69. The CARD Clinic in December 2005 performed a study comparing chest x-ray readings on 44 patients by CARD doctors, by ATSDR readers, and by Grace hired doctors. In the fall of 2005, the Grace Libby Medical Plan sent letters of denial to about a quarter of those enrolled in the plan. This was done through the plan administrator Health Network of America (HNA). The denials were based on chest x-ray readings by doctors

hired by the Grace Plan showing no asbestos disease. The Center for Asbestos Related Disease in Libby took the first 70 letters of denial brought in by patients, and compared the CARD readings against those by the U.S. Agency for Toxic Substances and Disease Registry (ATSDR), where available, and readings by outside doctors, where available. 44 of the 70 patients with Grace Plan denials had had chest x-rays read by the ATSDR in connection with screenings done in 2000 and 2001. See Peipins et al (2003). In 27 of 44 cases, the ATSDR confirmed the CARD finding of asbestos disease. Fourteen times the ATSDR readers on balance agreed with the Grace Plan hired doctors. There were three ties. In sum, the ATSDR was about 2x as likely to confirm the CARD readings. See Exhibit 15, Audit of HNA Denials and Downgrades of Severity of Disease (December 2005 revised).

Sixty-eight of the 70 patients denied by the Grace Plan had had chest x-rays and/or CT scans read by Dr. Steven Becker, radiologist at the St. John's Hospital in Libby. On 51 of 68 patients, Dr. Becker concurred with CARD. On the 17 where Dr. Becker did not concur, 16 of 17 CARD chest x-ray readings were confirmed by CT or other outside reading. On the one remaining, the CARD Clinic agrees that its initial reading was incorrect.

70. In 2007, an audit was done upon the chest x-ray readings by Dr.

David Weill, who was hired by W.R. Grace & Co. to read chest x-rays on 380 patients of the Center for Asbestos Related Disease. In many cases Dr. Weill read chest x-rays with dates on or near the dates of chest x-ray screenings done in Libby by the ATSDR. These chest x-rays were read by independent readers not hired by Grace or by the Libby Claimants. To investigate the question whether the ATSDR readers agreed more often with Grace's Dr. Weill or with the CARD Clinic treating physicians in Libby, on the issue of whether or not disease was present, a comparison study was performed. Results for the 59 patients, who are clients of McGarvey, Heberling, Sullivan & McGarvey, with reads by Dr. Weill, CARD and the ATSDR are tabulated on Exh. 19 attached. Results are summarized as follows:

11	All agreed (Weill, CARD, ATSDR)
26	ATSDR agreed with CARD
16	ATSDR agreed with Weill
2	form unreadable
4	tie 1/1 (1 ATSDR reader agreed with each)
59	total with ATSDR readings

The result is that where Grace's Dr. Weill and CARD disagreed, the ATSDR agreed with the CARD Clinic doctors 62% of the time (26/42), and agreed with Dr. Weill only 38% of the time (16/42). Regarding the 16 where the ATSDR agreed with Dr. Weill, followup chest x-rays or CT scans as read by independent radiologists confirmed the readings by the CARD Clinic doctors

on all 16 patients.

71. To compare CARD pulmonary function test (PFT) results to other pulmonary labs in northwest Montana, a study was undertaken. Certain patients who are clients of the law firm McGarvey, Heberling, Sullivan & McGarvey had independent medical exams (IMEs) or defense medical exams in the course of litigation. In Montana occupational disease cases, the State of Montana arranges IMEs called "panel exams." In addition, the defending insurance company in a Montana occupational disease case may arrange a defense medical examination. Also certain patients had had defense exams arranged for by the Burlington Northern Santa Fe Railroad.

All such IMEs were collected. IMEs with PFTs done within six months of a CARD PFT were selected. One was excluded as the hospital concluded it was a poor test. Comparison results are summarized on Exhibit 23 for the remaining 22 patients.

The lung function test measures of forced vital capacity (FVC), total lung capacity (TLC) and diffusion capacity (DLCO) were chosen as they are the three key indicators of severity in asbestos disease. Highest FVC before or after bronchodilator was used. Individual tabulations and underlying PFT records are attached on a CD titled "PFT Comparison Study 7/23/07." On Exhibit 23, "Percent difference" means the extent to which the independent pulmonary lab numbers were above or below the results obtained by the

CARD Clinic. The average difference was -3.68%, meaning that the CARD Clinic results were on the average about 3.68% higher than the independent pulmonary labs in Kalispell and Missoula. The difference is within the margin of error on lung function tests. The higher results obtained by the CARD Clinic lab may mean that technicians at the CARD Clinic were of higher competency than were those in the independent laboratories.

J. Other Observations

72. Blunting or obliteration of the costophrenic angle is a chest x-ray sign. The costophrenic angle is on the side of the chest x-ray at the lower end of the lung area. No pulmonologist would use blunting to determine a diagnosis of asbestos pleural disease. Nor would a practitioner use blunting to determine severity of asbestos pleural disease.

ILO (2000) includes blunting in its scoring definition for diffuse pleural thickening. Cases with diffuse pleural thickening and no blunting fall out of the ILO (2000) scoring system and are called out as pleural plaques. This is a false result. Diffuse pleural thickening may be present in the lower, middle, and/or upper lung zones, whether or not there is blunting in the distant costophrenic angle. ILO (2000) cites no medical literature in support of its new classification for diffuse pleural thickening. No blunting requirement appeared in the earlier version of the ILO Guidelines. Light and

Lee, Textbook of Pleural Diseases (2nd Ed. 2008), p.502, states that the ILO (2000) scheme is "not of much value clinically" for pleural disease.

In the CARD mortality study, of the patients who died of nonmalignant asbestos disease, only 43% (33/76) had blunting of a costophrenic angle.

Definitions of diffuse pleural thickening do not require blunting of the costophrenic angle. ATS (2004), p.707, states "diffuse thickening of the visceral pleura is not sharply demarcated . . . (it) extends continuously over a portion of the visceral pleura, often causing adhesions to the parietal pleura, involving fissures and obliterating the costophrenic angle." ATS (2004) does not include blunting in the diagnostic criteria for asbestos-related disease.

Similarly, the Frazer and Pare text, p.2804, defines diffuse pleural thickening as "with or without obliteration of the costophrenic sulci (56, 57)." The Rosenstock text, p.369, describes diffuse pleural thickening as "may involve costophrenic sulcus." McLoud (1985), p.14, studied 185 cases of diffuse pleural thickening and found that costophrenic angle obliteration "was not a consistent accompaniment of diffuse thickening." McLoud (1985), p.16, states that blunting was often found where diffuse pleural thickening was due to effusions, however, "it is rare when the appearance is due to confluent pleural plaques" (which was 25% of the 185 cases). Lilis et al (1986), in an analysis of 1,117 insulators found 23% with

measurable pleural thickening and only 13% with blunting.

Lilis et al (1991), abstract, states "the obliteration of the costophrenic angle(s), even with pleural fibrosis of limited extent, resulted in marked decrement in FVC percentage predicted." No data are presented indicating the extent of correlation. In any event, FVC is the standard measure of severity, not blunting. There is no suggestion by the authors that blunting could ever be applied to individual patients as a measure of severity of impairment. Lilis et al (1991) did not study lung function decrements in cases of pleural thickening without blunting, and presents no comment on severity of lung function impairment in cases without blunting. Moreover, Lilis (1991) only tests one of the three measures of severity of asbestos disease, FVC, TLC and DLCO. A patient may have severe impairment with severe shortness of breath, and only one of FVC, TLC or DLCO is in the severe range. Generally, with pleural thickening there is no correlation "between radiographic severity and longitudinal loss of lung function." Yates et al (1996), p.305.

73. ILO (2000) includes a minimum requirement of 3mm thickness in its scoring definition of "diffuse pleural thickening." There is no scientific basis for a requirement of 3mm thickness to constitute "diffuse pleural thickening." Without the 3mm minimum thickness, cases of pleural

thickening drop out of the ILO scoring system and are called pleural plaques. This is a false result. The earlier version of the ILO Guidelines did not have a 3mm minimum requirement for pleural thickening.

ATS (2004), Official Statement, p.707, notes that pleural thickening "ranges in thickness from less than 1mm up to 1cm or more." No minimum thickness is required for the diagnosis of asbestos-related disease under the ATS (2004) Official Statement diagnostic criteria.

Nor is thickness of pleural thickening a measure of the severity of impairment. As noted in the letter by the President of the American Thoracic Society, "diffuse pleural scarring can be associated with greatly diminished FVC regardless of the extent or thickness of the scarring or its bilaterality." Exhibit 18. As noted, with pleural thickening there is no correlation "between radiographic severity and longitudinal loss of lung function." Yates et al (1996), p.305. Thickness of pleural thickening was included in the pleural scoring system tested in Yates et al (1996). The 3mm requirement is contrary to standard practice in diagnosing asbestos pleural disease and contrary to the ATS (2004) criteria for diagnosis of asbestos-related disease.

In the CARD mortality study, 17% (13/76) of those who died of nonmalignant asbestos disease had less than a 3mm thickness in pleural

thickening.

The ILO (2000) requirement of 3mm minimum thickness to call out pleural thickening is dysfunctional and has no relation to the practice of medicine.

74. The National Institute for Occupational Safety and Health certifies "B Readers" who have passed a test on selected chest x-rays which are characteristic of certain scoring levels in the ILO (2000) classification system. The ILO (2000) scoring system for chest x-rays and the B Reader certification is for research purposes. As noted in Light and Lee, Textbook of Pleural Diseases, p. 502, the ILO system "is useful for epidemiology purposes but not of much use clinically." As noted above, the ILO system of classification is never used for diagnosis, and is of no value in the classification of severity of impairment in a particular patient. Lung function tests are the gold standard for measurement of functional impairment in individual patients. And, even lung function tests often do not correlate with degree of shortness of breath or degree of hypoxia.

The ILO classification system is not used in clinical practice in the Northwest United States. The number of B Readers has declined of late. There is no B Reader in Montana.

K. Grace/ACC Medical Criteria.

75. I have reviewed the medical criteria in the Grace/ACC Plan of Reorganization, Trust Distribution Procedures (TDP). The TDP category for "Severe Disabling Pleural Disease", p.26, note 7, "restricts diffuse pleural thickening to cases where there is associated blunting of the costophrenic angle." The TDP requires a finding of blunting of the costophrenic angle, before the patient is determined to have "diffuse pleural thickening." This is an incorrect definition of "diffuse pleural thickening."

Blunting or obliteration of the costophrenic angle is a chest x-ray sign. The costophrenic angle is on the side of the chest x-ray at the lower end of the lung area. No pulmonologist would use blunting to determine a diagnosis of asbestos pleural disease. Nor would a practitioner use blunting to determine severity of asbestos pleural disease.

ILO (2000) includes blunting in its scoring definition for diffuse pleural thickening. Cases with diffuse pleural thickening and no blunting fall out of the ILO (2000) scoring system and are called out as pleural plaques. This is a false result. Diffuse pleural thickening may be present in the lower, middle, and/or upper lung zones, whether or not there is blunting in the distant costophrenic angle. ILO (2000) cites no medical literature in support of its new classification for diffuse pleural thickening. No blunting requirement appeared in the earlier version of the ILO Guidelines. Light and

Lee, Textbook of Pleural Diseases (2nd Ed. 2008), p.502, states that the ILO (2000) scheme is "not of much value clinically" for pleural disease.

In the CARD mortality study, of the patients who died of nonmalignant asbestos disease, only 43% (33/76) had blunting of a costophrenic angle. Attached as Exhibit 17 is a letter dated February 9, 2005, titled "Preliminary Report of 79 chest x-rays reviewed relative to the Asbestos Injury Resolution Act of 2005." This study was done in response to a similar requirement of blunting of the costophrenic angle in a draft of the asbestos bill. It is my understanding that this requirement was removed from the asbestos bill in the special provisions for Libby, as reported out of the Senate Judiciary Committee on May 26, 2005. As noted on the preliminary report in 2005, only 22 of 79 (28%) patients had blunting of the costophrenic angle.

Definitions of diffuse pleural thickening do not require blunting of the costophrenic angle. ATS (2004), p.707, states "diffuse thickening of the visceral pleura is not sharply demarcated . . . (it) extends continuously over a portion of the visceral pleura, often causing adhesions to the parietal pleura, involving fissures and obliterating the costophrenic angle." ATS (2004) does not include blunting in the diagnostic criteria for asbestos-related disease.

Similarly, the Frazer and Pare text, p.2804, defines diffuse pleural thickening as "with or without obliteration of the costophrenic sulci (56,

57)." The Rosenstock text, p.369, describes diffuse pleural thickening as "may involve costophrenic sulcus." McLoud (1985), p.14, studied 185 cases of diffuse pleural thickening and found that costophrenic angle obliteration "was not a consistent accompaniment of diffuse thickening." McLoud (1985), p.16, states that blunting was often found where diffuse pleural thickening was due to effusions, however, "it is rare when the appearance is due to confluent pleural plaques" (which was 25% of the 185 cases). Lilis et al (1986), in an analysis of 1,117 insulators found 23% with measurable pleural thickening and only 13% with blunting.

Lilis et al (1991), abstract, states "the obliteration of the costophrenic angle(s), even with pleural fibrosis of limited extent, resulted in marked decrement in FVC percentage predicted." No data are presented indicating the extent of correlation. In any event, FVC is the standard measure of severity, not blunting. There is no suggestion by the authors that blunting could ever be applied to individual patients as a measure of severity of impairment. Lilis et al (1991) did not study lung function decrements in cases of pleural thickening without blunting, and presents no comment on severity of lung function impairment in cases without blunting. Moreover, Lilis (1991) only tests one of the three measures of severity of asbestos disease, FVC, TLC and DLCO. A patient may have severe impairment with

severe shortness of breath, and only one of FVC, TLC or DLCO is in the severe range. Generally, with pleural thickening there is no correlation "between radiographic severity and longitudinal loss of lung function." Yates et al (1996), p.305.

Blunting cannot be an element in the diagnosis of diffuse pleural thickening. Nor can blunting be a measure of severity of pleural disease. Lung function tests are the measure of severity of asbestos disease. Rosenstock text, p.370. Miles et al (2008), "Clinical consequences of asbestos-related diffuse pleural thickening, a review," p.6, states: "Few longitudinal studies exist, but these have found no correlation between radiographic severity and longitudinal loss of lung function." The use of blunting as a necessary condition to a diagnosis of diffuse pleural thickening is scientifically arbitrary. Likewise, any use of blunting to determine severity of functional impairment is scientifically arbitrary.

76. The Trust Distribution Procedures (TDP) category for "Severe Disabling Pleural Disease," p.26, requires thickness of "at least width 'a'" for diffuse pleural thickening. "Width 'a'" is defined "based on definitions as set forth in the 2000 revision of the ILO classification." ILO (2000), p.7, defines width 'a' as 3-5mm. The TDP requires a finding of thickness of width "a" (3mm) before the patient is determined to have "diffuse pleural thickening."

This is an incorrect definition of "diffuse pleural thickening." No pulmonologist would use a minimum 3mm thickness as a necessary condition to a diagnosis of diffuse pleural thickening. No practitioner would use a minimum 3mm thickness to determine severity of asbestos pleural disease.

ILO (2000) provides a scoring system for chest x-rays for research purposes. Light and Lee, *Textbook of Pleural Diseases* (2nd Ed. 2008), p.502, states that the ILO (2000) scheme is "not of much value clinically" for pleural disease. ILO (2000) includes the minimum 3mm thickness in its scoring definition of diffuse pleural thickening (DPT). Cases with DPT and no 3mm thickness fall out of the ILO (2000) scoring system, and are called out as pleural plaques. This is a false result. ILO (2000) cites no medical literature in support of its new classification requiring the 3mm minimum. There was no minimum thickness in the earlier version of the ILO Guidelines. Width "a" was 0-5mm.

In the CARD mortality study, of the patients who died of nonmalignant asbestos disease 83% (63/76) had the 3mm minimum thickness on chest x-ray. This is consistent with the clinical observation that many patients have died with thin diffuse pleural thickening, which is extensive. On CT scan, more pleural thickening is seen. Many more patients would qualify with the

3mm thickness if CT scans were used.

Similarly, the measurements for the 2005 CARD study of 79 sets of chest x-rays show that about 38% of the patients were excluded by a requirement of a minimum 3mm thickness. Exh. 17.

The ATS (2004) Official Statement diagnostic criteria for asbestos-related disease includes no requirement of a minimum thickness for diffuse pleural thickening. No diagnostic definition of diffuse pleural thickening includes a requirement of a minimum thickness. In fact, ATS (2004), Official Statement, p.707, notes that pleural thickening "ranges in thickness from less than 1mm up to 1cm or more."

Nor is thickness of pleural thickening a measure of the severity of impairment. As noted in the letter by the President of the American Thoracic Society, "Diffuse pleural scarring can be associated with greatly diminished FVC regardless of the extent or thickness of the scarring or its bilaterality." Exh. 18. As noted above, with pleural thickening there is no correlation between "radiographic severity and longitudinal loss of lung function." Yates et al (1996), p.305.

The use of 3mm thickness as a necessary condition to diagnosis of diffuse pleural thickening is scientifically arbitrary. Likewise, any use of the 3mm thickness to determine severity of functional impairment is scientifically

arbitrary.

77. The TDP category for "Severe Disabling Pleural Disease," p.26, defines diffuse pleural thickening as "at least extent '2' . . . based on definition set forth in the 2000 revision of the ILO classification." ILO (2000), p.7, states that extent "2 = total length exceeding one-quarter and up to one-half of the projection of the lateral chest wall." The TDP requires a finding of "extent > 25%" before the patient is determined to have "diffuse pleural thickening." This is an incorrect definition of "diffuse pleural thickening." No pulmonologist would use "extent > 25%" as a necessary condition to a diagnosis of diffuse pleural thickening. No practitioner would use "extent > 25%" to determine functional severity of asbestos pleural disease.

In the CARD mortality study, of the patients who died of nonmalignant asbestos disease 17% (62/75) did not have extent of pleural thickening greater than 25% of the chest wall. This is consistent with clinical observation that many patients have died of pleural disease without the "extent > 25%," especially when observed only on a chest x-ray. Extent of pleural thickening is better seen on CT scan. Many more patients would qualify with "extent > 25%" if CT scans were used.

The ATS (2004) Official Statement diagnostic criteria for asbestos-

related disease includes no requirement of "extent > 25%" for diagnosis of diffuse pleural thickening. Nor is the extent of pleural thickening a measure of severity of impairment. As noted by the President of the American Thoracic Society, "diffuse pleural scarring can be associated with greatly diminished FVC regardless of the extent or thickness of the scarring or its bilaterality." Exh. 18. As noted above, with pleural thickening there is no correlation "between radiographic severity and longitudinal loss of lung function." Yates et al (1996), p.305.

The use of "extent > 25%" as a necessary condition to a finding of diffuse pleural thickening is scientifically arbitrary. Likewise, any use of "extent > 25%" to describe severity of functional impairment is scientifically arbitrary.

78. The TDP excludes cases of unilateral disease. The TDP category for "Severe Disabling Pleural Disease" category requires that diffuse pleural thickening be "one component of a bilateral nonmalignant asbestos-related disease." Most Libby patients who have unilateral disease later develop bilateral disease. In the CARD mortality study group of 79 nonmalignant asbestos disease deaths, 1% (1/79) had unilateral disease. Exh. 7.

Asbestos pleural disease may be unilateral and severe. The President of the American Thoracic Society states: "Diffuse pleural scarring can be

associated with greatly diminished FVC regardless of the extent or thickness or scarring or its bilaterality." Exh. 18.

Diffuse pleural thickening is often unilateral. A requirement in the TDP excluding unilateral cases is scientifically arbitrary, as practicing physicians may treat unilateral cases which are just as severe as bilateral cases.

Miles et al (2008), "Clinical Consequences of Asbestos-Related Diffuse Pleural Thickening: A Review," p.5, states "Approximately one-third of cases of DPT are unilateral." The Rosenstock text, p.369, states that pleural thickening is "usually bilateral." The Fishman text, p.943, states that pleural thickening can be "either bilateral or unilateral."

Other categories in the TDP, pleural disease levels 1, 2 and 3, also require bilateral pleural disease, excluding unilateral disease. These classifications too are scientifically arbitrary.

The ILO (2000) classifications recognize unilateral disease. Its pleural thickening scoring system, p.7, refers to "an obliterated costophrenic angle" implying that one angle is sufficient. Similarly the scoring system, p.7, for diffuse pleural thickening records measurements "separately for the right and left side," implying that a single 3mm width in a single "extent > 25%" will suffice. However, the TDP does not follow ILO (2000), as the TDP excludes unilateral disease.

The exclusion of unilateral disease is scientifically arbitrary and has no relationship to the treatment of asbestos disease patients.

79. The TDP omits diffusion capacity (DLCO) as a measure of severity. The TDP, p. 26, for "Severe Disabling Pleural Disease" Level 4B, requires Total Lung Capacity (TLC) or Forced Vital Capacity (FVC) under 65. Of all lung function tests, the three most important in asbestos disease are FVC, TLC, and diffusion capacity. The Fishman text, p. 950, states "The characteristic pulmonary function changes of asbestosis are a restrictive impairment with a reduction in lung volumes (especially FVC and TLC), decreased diffusion capacity, and arterial hypocemia." ATS (2004) Official Statement adopted the above quoted statement at p. 697. The 2005 Public Citizen comment by Dr. Michael Harbut, Dr. Philip J. Landrigan, Dr. Alan C. Whitehouse and Dr. L. Christine Oliver states that DLCO is "essential to determine how badly a person's lungs are impaired." Exh. 16. The TDP's omission of diffusion capacity as a measure of severity is not consistent with the medical literature, or clinical practice.

Diffusion Capacity (DLCO) is a particularly important indicator of the severity of impairment in the Libby asbestos disease patients. In some cases, there is significant asbestos disease on the chest x-ray and only the DLCO is reduced, not the FVC or TLC. Some patients with severe shortness

of breath are severe only in the DLCO defect. In Whitehouse (2004), p. 224, 76% of the 123 patients had progressive loss of lung function. Losses were about the same for each of FVC, TLC and DLCO at 2-3% per year.

In the CARD mortality study, among those who died of nonmalignant asbestos disease, 44% (30/68) had only DLCO under 65 (out of FVC, TLC and DLCO). These patients were all severe and are now dead. Yet, none would qualify as a "severe pleural" under the TDP because they did not happen to have FVC or TLC under 65. In terms of clinical medicine, the exclusion is scientifically arbitrary.

80. The TDP excludes those with an obstructive component to their asbestos pleural disease. TDP, p. 26, for "Severe Disabling Pleural Disease", Level 4B, and for moderate "pleural disease," Level 3, requires an "FEV_1/FVC ratio > 65%." As discussed above in § H Obstructive Defect from Asbestos Related Disease, an obstructive pattern is common with ARD, particularly in the Libby cohort. To exclude those with significant Asbestos Pleural Disease and an obstructive component would be scientifically arbitrary.

The Fishman text, p. 604, states: "The hallmark of the obstructive pattern is a reduction in the FEV_1/FVC percentage. . .Typically, all three lung volumes, residual volume, FRC, and TLC are increased." Here, increased means abnormal, or over 120% of predicted. A mild reduction in FEV_1/FVC

ratio may be seen as a ratio of 60-70. Markowitz et al (1997), p. 102, uses a ratio of 70 as normal for ages under 60 and a normal of 65 for those 60 or older.

The problem is that smoking disease may also cause reduction in the ratio, and there may be an interest in excluding persons with minimal asbestos disease and predominately smoking disease. A more scientifically sound approach would be to exclude those with a ratio under 65 and TLC abnormally high (over 120). Of the CARD mortality study nonmalignant deaths, 8% (6/71) of those with full lung function tests had a total lung capacity over 120. As I understand it, these six would go to individual review.

No requirement of an FEV_1/FVC ratio over 65 is included as a necessary condition to the diagnosis of asbestos-related disease in the ATS (2004) Official Statement on standard practice in diagnosis of ARD. No diagnostic definition of ARD includes a requirement that the FEV_1/FVC ratio be over 65. Excluding those patients with a ratio under 65 from the pleural disease classification is scientifically arbitrary. The interest in excluding those with smoking disease and no significant asbestos disease is understood. This may be properly accomplished through a requirement that total lung capacity (TLC) be abnormally high (over 120).

81. The data and other information considered in forming the above opinions and observations include:

a. Patient information and observations as treating physician.

b. All information referenced above.

c. Medical literature on asbestos disease.

d. Consultations with Dr. Arthur Frank, and his expert report.

e. Consultations with epidemiologist Dr. Craig Molgaard.

f. A listing of cases with trial or deposition testimony is attached.

Compensation is at the rate of $350 per hour.

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DATED this 29th day of December, 2008.

Alan C. Whitehouse MD, FCCP

List of Exhibits to Expert Report by Dr. Alan C. Whitehouse

1. Whitehouse, A.C., Curriculum vitae.

2. "Asbestos-Related Pleural Disease Due to Tremolite Associated with Progressive Loss of Lung Function: Serial Observations in 123 Miners, Family Members, and Residents of Libby, Montana," Am J Ind Med 46:219-225 (2004).

3. "Environmental Exposure to Libby Asbestos and Mesotheliomas," Whitehouse (2008) Am J Ind Med.

4. Client Sort by Exposure (Community/Family Member/Worker) (CARD patients only).

5. Dead in 123 patients in Whitehouse (2004).

6. Chart "Case # Type of Progression" with x-rays on CD attached.

7. Chart, "Summary of Mortality Study Disease Percentages." Mortality data are on a CD titled "CARD Mortality Study Spreadsheets," containing spreadsheets on 79 ARPD deaths, 37 cancer deaths, and death certificate information for 116 deaths. "116 patient medical records and death certificates in CARD Mortality Study" is a separate CD. Patient information on non clients is redacted.

8. Printout, "Libby Claimants on Oxygen (CARD patients only)," 5/28/08 and "Libby Claimants with FVC, TLC or DLCO < 60 (CARD patients only)," 5/28/08.

9. Chart "Mesothelioma Cases Due to Exposure to Libby Asbestos." Documents supporting the chart are on the CD titled "Meso Cases Libby 7/30/07."

10. Chart Deceased clients of MHSM (death date order) and chart of Deceased Clients of LSK (death date order).

11. Charts 035a "Workers with Disease - 1969," 035b "Workers with Disease - 1975," and 035c "Workers with Disease - 1976."

12. Cookson (1986), Fig. 1. Chart W-2 "Years since first exposed."

13. Chart, Studies on Radiographic Progression of Asbestos Disease.

14. Chart, Workers Dead from Asbestos Disease (2000 study).

15. Audit of HNA Denials and Downgrades of Severity of Disease (December 2005), and chart of data with non-Libby Claimants deleted.

16. Public Citizen, "Leading Medical Experts Fault Arbitrary, Outdated Medical Criteria in Asbestos Bill," (May 2005).

17. Letter dated February 9, 2005, titled "Preliminary Report of 79 Chest X-rays Reviewed Relative to the Asbestos Injury Resolution Act of 2005, with attached Log of Patients Criteria Study, FVC and on Oxygen (non-Libby Claimants' names are blacked out.)

18. Letter by the President of the American Thoracic Society.

19. Chart, "Weill comparison to ATSDR and CARD." Documents supporting the chart are on the CD titled "Weill Comparison 7/23/07."

20. W.R. Grace & Co., "Source Emissions - Results of Surveys 1975."

21. Chart, "Libby Cohort Deaths Per Source."

22. W.R. Grace & Co., memo, McCaig to Walsh 9/17/85.

23. Chart, "Center for Asbestos Related Disease, Libby Pulmonary Function Test (PFT) Comparison with Independent Medical Exam PFTs." Documents supporting the chart are on the CD titled "CARD PFT Comparison with IME PFTS 7/23/07."

24. Listing of cases with deposition or trial testimony.

LIBBY STUDIES

AUTHOR/ EDITOR	PUB DATE	TITLE OF ARTICLE/TEXT
Amandus	1986	The Morbidity and Mortality of Vermiculite Miners and Millers Exposed to Tremolite-Actinolite
Amandus	1987	Prevalence of Radiographic small opacities in vermiculite miners
Amandus I	1987	The Morbidity and Mortality of Vermiculite Miners and Millers Exposed to Tremolite-Actinolite: Part I. Exposure Estimates
Amandus II	1987	The Morbidity and Mortality of Vermiculite Miners and Millers Exposed to Tremolite-Actinolite: Part II. Mortality
Amandus III	1987	The Morbidity and Mortality of Vermiculite Miners and Millers Exposed to Tremolite-Actinolite: Part III. Radiographic Findings
Atkinson	1982	Interim Final Report
EPA	1982	Collection Analysis and Characterization of Vermiculite Samples for Fiber Content and Asbestos Contamination
EPA	1991	Health Assessment Document for Vermiculite
Hart	2007	Evaluation of Asbestos Exposures during Firewood-Harvesting Simulations in Libby, MT USA - Preliminary Data
Horton	2008	Select Mortality and Cancer Incidence Among Residents in Various U.S. Communities that Received Asbestos-Contaminated Vermiculite Ore from Libby, Montana
Lockey	1984	Pulmonary Changes after Exposure to Vermiculite Contaminated with Fibrous Tremolite
McDonald	1986	Cohort Study of Mortality of Vermiculite Miners Exposed to Tremolite
McDonald	1986	Radiological Survey of Past and Present Vermiculite Miners Exposed to Tremolite
McDonald	1988	Health of Vermiculite Miners Exposed to Trace Amounts of Fibrous Tremolite
Whitehouse	2004	Asbestos-Related Pleural Disease Due to Tremolite Associated with Progressive Loss of Lung Function: Serial Observations in 123 Miners, Family Members, and Residents of Libby, Montana
Whitehouse	2008	Environmental Exposure to Libby Asbestos and Mesotheliomas

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